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Theoretical and practical significance of parallel assays of serum lactic acid, pH and blood gases in mothers and neonates at birth

D. Kalinkov*, H. Schachinger, R. Buchholz***

* Department of Obstetrics and Gynecology, University of Marburg, W. Germany
** Department of Pediatrics, The Free University of Berlin, W. Germany

1 Introduction

Anaerobic glycolysis causes metabolic acidosis with accumulation of lactate. The significance and the usefulness of lactate assays in the fetus and the mother during and after delivery is well documented [1, 2, 8, 9, 17, 20]. Using the Lactate Analyser 640 from HOFFMANN LA ROCHE, we did systematic lactate level assays at delivery. The object of this study was to gather further information on the acid-base conditions in the newborn and the causes of fetal acidosis.

2 Materials

In 126 vaginal and 32 abdominal deliveries, three blood samples were taken immediately after delivery:

- maternal blood from the radial artery;
- neonatal blood from the umbilical vein and artery,

No signs of fetal distress were present in 98 of the vaginal deliveries and 13 of the primary cesarean sections, whereas 28 of the vaginal deliveries involved this complication and 19 cesarean sections were performed because of or at least in the presence of fetal distress.
In all three blood samples, pH and blood gas status and lactate levels were determined immediately.

Curriculum vitae

DIMITRE KALINKOV *was born in 1948, graduated from the Louis Pasteur University of Strasbourg (F) in 1973. From 1973 to 1975 research in the field of metabolic acidosis in neonatology at the Institute for Human Physiology and at the 2nd Dept, of Obstetrics and Gynecology of the Medicine University of Strasbourg. Since 1976 he has been working in the Dept. of Obstetrics and Gynecology of the Philips University of Marburg. He qualified as a specialist in Obstetrics and Gynecology in 1978. Since 1979 he has held the position of Assistant Professor at the University of Marburg. Main field of research interest; metabolic acidosis in pregnanture labour, urinary incontinence and early recognition of ovarian carcinoma.*

3 Methods

All data on the acid-base status were obtained on the Autocal pH/Blood Gas Analyser 613 from Instrumentation Laboratory.
Older spectrophotometric methods of assaying lactate were too time consuming and elaborate for use in acute situations [3, 6, 10]. We used the HOFFMANN LA ROCHE Lactate Analyser 640 [4, 11, 13]. Maternal arterial lactate levels (L_M) and newborn lactate levels from the umbilical vein (L_V) and artery (L_A) were determined.

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Initially, it was not clear whether the lactate measured by the method suggested by HOFFMANN LA ROCHE come from the plasma or the intraerythrocytic fluid. Since the lactate levels were determined without consideration of varying hematocrit values [16], we did a few preliminary studies on the method itself.

In twenty cord blood samples (mean hematocrit – 50%) lactate was assayed:

A. in plasma following immediate centrifugation at 0 °C (L_Z);

B. in whole blood following immediate hemolysis (L_H), Lactate was measured from the total fluid components of the blood, i.e. plasma and intraerythrocytic fluid [12];

C. in whole blood according to the HOFFMANN LA ROCHE method (L_R), whereby assays were done on a mixture of plasma and the fluid content from red blood cells hemolysed by manipulation of the sample (shaking etc.).

The lactate measurements were repeated 15 and 60 minutes after the samples had been taken (Fig. 1).

Centrifugation was the most elaborate method. For obvious technical reasons the first value was not available until 15 minutes after sampling.

After hemolysis the initial value L_H exceeded whole blood values without hemolysis L_R by 15%. It was absolutely independent of time, since glycolysis was blocked. This method is useful when the Lactate Analyser apparatus is not immediately accessible and when the fast result are not essential.

	5' POST PARTUM	15' POST PARTUM	60' POST PARTUM
L_Z	-	3,95	4,13
L_H	3,25	3,22	3,22
L_R	2,83	3,46	5,22

Fig. 1. Parallel assays of lactate contents in plasma after centrifugation at 0 °C (L_Z) and in whole blood after hemolysis (L_H) or after primary dilution according to the method of HOFFMANN LA ROCHE (L_R), 5, 15 and 60 minutes after blood sampling, i.e., after delivery.

We preferred the method of HOFFMANN LA ROCHE, in which lactate levels are measured right after blood sampling. Since a linear increase in the lactate velues with time was observed, the initial level (L_{Ro}) can be computed on the basis of the value at time t in minutes after sampling using the following formula:

$$L_{Ro} = L_{Rt} - 0.04\ t.$$

4 Results

In Fig. 2 our measurements are summarized for a group of ten patients in the 40th week of pregnancy before commencement of labor, in 111 births without fetal distress, and in 47 births with signs of fetal distress.

Our pH and blood gas data are in agreement with those reported in the literature (cf. a review of 24 authors [9]).

The lactate levels in normal deliveries however, were about 20% lower than those previously reported, which had been assayed by spectrophotometry [8]. More than three hours were necessary for measurement. The higher levels suggest that glycolysis continues even in denatured blood.

Lactate levels in neonates born without fetal distress were lower than those in the mother, whereas those in cases of fetal distress exceeded maternal values.

During induction of anesthesia for primary cesarean section, lactate in the mother increased from 0.40 mmol/l to 1.66 mmol/l. Similar observations were also made in gynecological operations. The only explanation available lies in artificial respiration during preparation for laryngeal intubation. Experiments have shown that mechanical assistance produces hypocapnia and a bicarbonate deficit, resulting in metabolic acidosis [5]. Lactate and pyruvate levels increase concomitantly.

Our group of cesarean section patients also showed hypocapnia. It is interesting that hypocapnia is even more evident during vaginal delivery, but does not occur acutely. Maternal lactate acidosis due to hypocapnia in vaginal deliveries can be compensated, which is impossible in the short time between intubation and delivery of the child by cesarean section.

			pH	PO_2 TORR	PCO_2 TORR	LACTAT MMOL/L
A	40th. gestation week	n = 10	7.40	91	38	0.40
B	NORMAL VAGINAL DELIVERIES N = 98	UV	7.33	26	37	1.98
		UA	7.25	15	48	1.80
		MOTHER	7.41	103	26	2.14
	PRIMARY CESAREAN SECTIONS N = 13	UV	7.31	29	35	1.17
		UA	7.24	20	40	1.09
		MOTHER	7.30	226	32	1.66
C	VAGINAL DELIVERIES WITH FETAL DISTRESS N = 28	UV	7.21	16	47	2.31
		UA	7.12	10	64	2.98
		MOTHER	7.34	92	28	2.40
	CESAREAN SECTIONS FOR FETAL DISTRESS N = 19	UV	7.18	16	50	3.11
		UA	7.10	9	65	3.92
		MOTHER	7.27	185	30	2.89

Fig. 2. pH, pO_2, pCO_2 and lactate mean values in arterial blood from ten patients in the 40th week of gestation before labor (A), at delivery in the umbilical vein (UV), in the umbilical artery (UA) and in maternal arterial blood in 98 vaginal and 13 abdominal deliveries without fetal distress (B) and in 28 vaginal and 19 abdominal deliveries with fetal distress (C).

5 Discussion

The relationships among certain parameters struck us as more interesting than the absolute data.

In vaginal deliveries (Fig. 3) the maternal arterial lactate level (L_M mmol/l) is closely correlated to the duration of the late second stage (t minutes):

$$L_M = 1.807 + 0.072\ t.$$

Muscular exertion on the part of the mother is enough to account for this.

In contrast, in the absence of fetal distress, maternal and neonatal lactate levels are closely correlated (Fig. 4):

$$L_V = 0.16 + 0.64\ L_M.$$

This suggests that:

- firstly, maternal lactate concentrations have an effect on those of the child, i.e., a maternal – fetal "infusion – acidosis" is conceivable;
- secondly duration of the late second stage affects the lactate concentration in cord blood.

The close correlation between arterial and venous cord blood lactate concentrations in deliveries with or without fetal distress is noteworthy in this context (Fig. 5).

Furthermore, we observed a relationship between the lactate level in venous cord blood (L_V mmol/l) and the condition of the neonate at birth, as measured by the APGAR score be expressed as follows for the normal lactate range from 1.50 to 6.50 mmol/l:

$$A_1 = 12.8 - 1.9\ L_V.$$

Clinically, it is desirable that the neonate have an APGAR score of 7 or better. In the neonate, lactate levels of 3 mmol/l should not be exceeded (Fig. 6). Such lactate levels in the newborn are observed when a maternal concentration of 4 mmol/l is reached (Fig. 4), i.e. after a late second stage lasting 25 minutes (Fig. 3).

In everyday obstetric practice, this means that the lactate level of the mother should be determined after six bearing down pains. In venous blood, concentrations of op to 4.20 mmol/l can

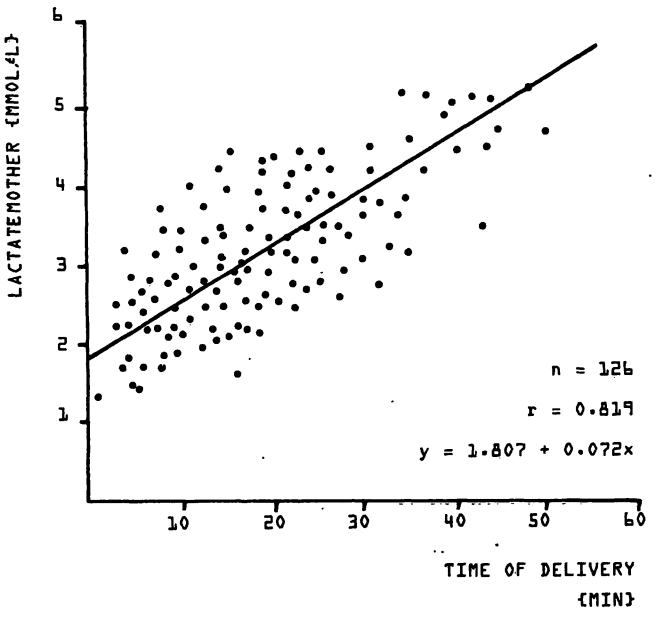

Fig. 3. Relation between duration of late second stage (min.) and maternal arterial lactate value (mmol/l). (N = 126).

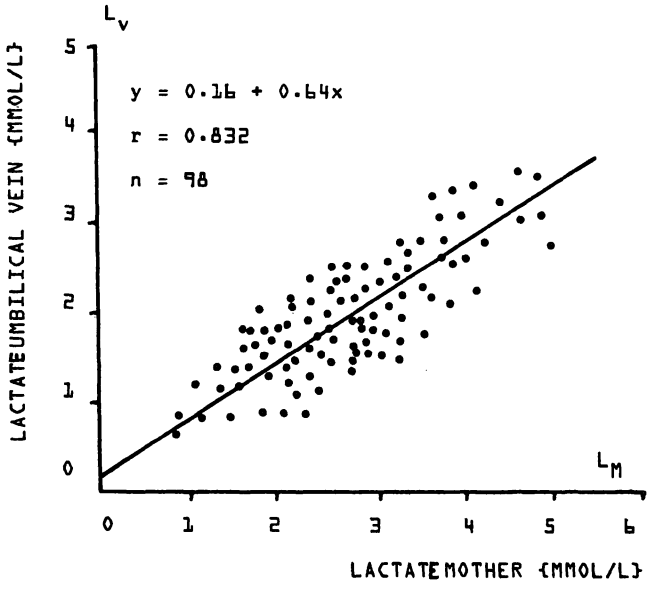

Fig. 4. Relation between maternal arterial and umbilical venous lactate values after normal vaginal delivery (mmol/l). (N = 98).

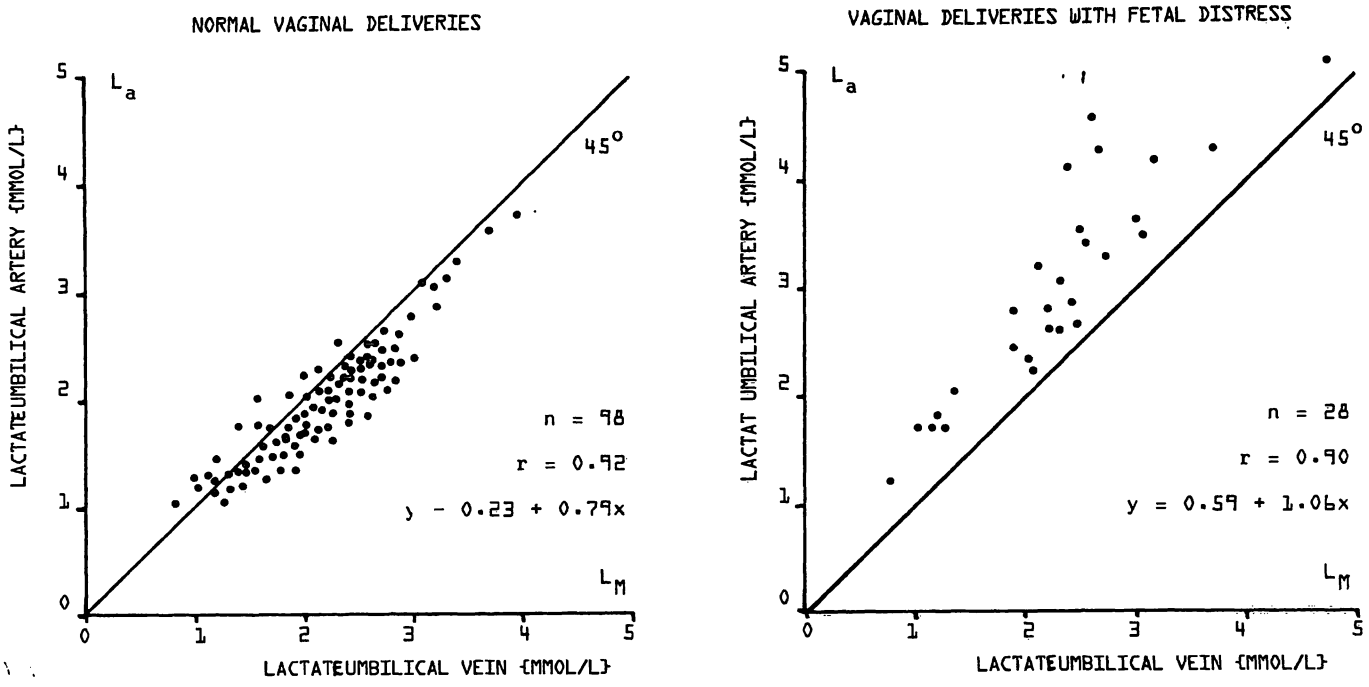

Fig. 5. Relation between arterial and venous umbilical lactate values in normal deliveries and in those with fetal distress.

be tolerated. Even in the absence of cardiotocographic signs of fetal distress, operative termination of delivery is indicated after ten bearing down pains [10, 15].

Lactate concentrations from maternal arterial and cord blood permit retrospective analysis of the causes of fetal acidosis when it occurs. Our data could be arranged in four groups according to etiology (fig. 7):

– Situation 1 is representative of a delivery with a short late second stage, usually in a multipara with a fetus of normal size. It was observed in 34 of our 98 vaginal deliveries without fetal distress and in 6 of the 13 primary cesarean sections;

– Situation 2 was the most frequent, occurring in 64 of our 98 vaginal deliveries and 4 of 13 primary cesarean sections. Typical examples of

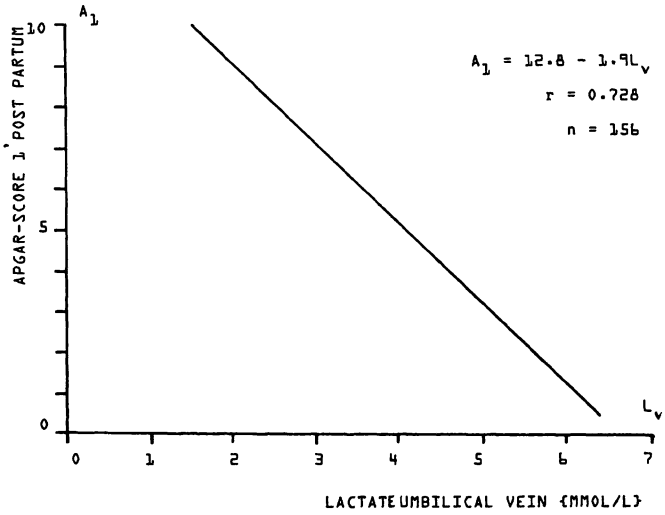

Fig. 6. Relation between umbilical venous lactate value (mmol/l) and APGAR score of the newborn 1 minute after delivery (N = 156).

1.	$L_a > L_v$ MOST $L_M > L_v$: (ALL VALUES < 3 MMOL/L)	NORMAL DELIVERY
2.	3 MMOL/L $> L_M > L_v > L_a$:	"INFUSED ACIDOSIS" WITHOUT CLINICAL REPERCUSSION
3.	$L_M > L_v > L_a >$ 3 MMOL/L :	FETAL DISTRESS IN CAUSE OF "INFUSED ACIDOSIS"
4.	$L_a > L_v >$ 3 MMOL/L $> L_M$:	PRIMARY FETAL DISTRESS

Fig. 7. Clinical and etiological classification of deliveries according to the relation between maternal arterial and umbilical venous or arterial lactate levels.

this situation are deliveries in primiparas with a long second stage, large fetus or pathological presentation:

– Situation 3 was observed in 16 of the 28 vaginal deliveries with fetal distress. These children would have been better off if the birth had been terminated of an operative procedure. We saw late second stages lasting more than 40 minutes in primiparas. One must consider which risk weighs more heavily, that of complications caused by extraction with forceps or vacuum or that of "infusion acidosis". We believe that the experienced obstetrician should tend to prefer operative delivery.
The lactate levels in 3 of the 13 primary cesarean sections also fit the pattern of situation 3. Excessive and unnecessary hyperventilation during induction of anesthesia was clearly the cause of fetal distress in every fourth primary section. Maternal pO_2 levels in excess of 230 Torr were observed, which were of no use to the fetus and only served to prolong intubation. Brief hyperventilation is thus more appropriate, whereby pO_2 levels of only 150–180 Torr are produced; this should be followed by quick intubation without further hyperventilation.

– Situation 4 was observed in 12 of 28 vaginal and 17 of 19 abdominal deliveries involving fetal distress.

In the remaining two sections maternal concentrations rose to more than 3 mmol/l, since the decision to operate was not made until the patient had gone through 30 minutes of bearing down labor without progress. In one case arterial cord blood lactate level was 11.03 mmol/l, corresponding to a maternal lactate level of 6.71 mmol/l. In this case a cesarean section was done because of complicated transverse lie. Surprisingly, the only abnormal CTG findings were Dip I's, which were observed for more than two hours. The neonate had an APGAR score of 1 one minute after delivery. Following intensive reanimation, the APGAR score was still only 7 after 10 minutes. Ten hours later, the lactate concentration in arterial neonatal blood was still highly pathologic at 4.12 mmol/l; the fetal heart rate was still practically silent.

Parallel assay of Lactate level, pH and blood gases in the mother and in arterial and venous cord blood permit:

– exact assessment of the condition of the neonate at birth and
– recognition of the etiology of any fetal acidosis present.

Of practical use are two additional points:

– more than 8 bearing down pains during a normal delivery can involve a threat of "infusion acidosis" in the fetus;
– induction of full anesthesia before cesarean section should be preceded by as brief a hyperventilation phase as possible.

Summary

Theoretical and practical significance of parallel assays of serum lactic acid, pH and blood gases in mothers and neonates at birth.
After preliminary studies of the lactate measuring method itself for assessment and specification of the blood components in which lactate was measured, we assayed maternal arterial (L_m) and cord blood lactate concentrations (L_a – umbilical artery and L_v – umbilical vein) in 126 vaginal and 32 abdominal deliveries, using the Lactate Analyser 640 from HOFFMANN LA ROCHE. The results were correlated to fetal outcome (CTG findings, APGAR scores), to duration of the late second stage, and to the usual blood gas and acid-base parameters.
We drew the following conclusions from our studies:

1. – there is a close correlation between maternal and fetal lactate levels in the absence of fetal distress;
2. – maternal lactate correlates well to the duration of late second stage;
3. – when the late second stage is short, the lactate concentration gradient from fetus to mother is usually maintained, or only small lactate infusions occur. The fetal outcome at delivery is thus not endangered;
4. – when the late second stage is long, the maternal lactate level is higher, permitting a maternal-fetal infusion acidosis; it appears after only 6 to 8 bearing down pains and can produce fetal distress after about ten bearing down pains;
5. – in the presence of primary fetal distress, fetal lactate levels are already excessive, so that even a long late second stage cannot produce an infusion acidosis because of the concentration gradient;
6. – an increase of maternal arterial lactate levels of about 1.2 mmol/l always accompanies induction of full anes-

thesia. Its etiology is discussed. This increase can cause infusion acidosis in primary cesarean sections, but not in sections due to of fetal distress.
Two practical conclusions can be derived from our studies:
– firstly, even when CTG-patterns remain normal, delivery should be terminated with an operative procedure after 8 bearing down pains at the most;
– secondly, hyperventilation preceding intubation anesthesia for cesarean section should be kept as short as possible to prevent metabolic infusion acidosis.

Keywords: Fetal distress, maternal-fetal infusion acidosis, lactate, late second stage, metabolic acidosis.

Zusammenfassung

Theoretische und praktische Bedeutung der parallelen Laktat-, pH- und Blutgasbestimmung bei Mutter und Kind zum Zeitpunkt der Geburt.
Nach einigen meßmethodischen Voruntersuchungen die zur genauen Definition der Blutmeßkomponente dienten, wurden bei 126 vaginalen und 32 abdominellen Entbindungen mütterliche arterielle (L_M) und kindliche Nabelschnurblutaktatkonzentrationen (L_a), in der Nabelarterie und L_v, in der Nabelvene) mit dem Laktatanalysator 640 der Firma Hoffmann La Roche gemessen. Die Ergebnisse wurden in Relation zum kindlichen Zustand (CTG-Befunden, APGAR-Werten), zur Dauer der Austreibungsphase und zu den routinemäßig bestimmten Blutgas- und Säure-Basenwerten gesetzt.
Es konnten folgende Schlußfolgerungen gezogen werden:
1. Es besteht ein enger Zusammenhang zwischen mütteröichem und kindlichem Laktatspiegel, wenn kein „fetal distress" vorliegt;
2. Der mütterliche Laktatspiegel korreliert eng mit der Austreibungsdauer.
3. Bei kurzer Austreibungsdauer bleibt das Laktatkonzentrationsgefälle meist in Richtung von kindlichen zu mütterlichen Werten, oder es werden nur geringe „Laktatinfusionen" erreicht. Somit kann hier der kindliche Zustand bei Geburt nicht beeinträchtigt werden;
4. Bei langer Austreibungsdauer überwiegt der mütterliche Laktatspiegel und es kann eine mütterlich-fetale „Infusionsazidose" entstehen. Diese tritt bereits nach 6 bis 8 Preßwehen auf und führt nach ca 10 Preßwehen zu einem „fetal distress".
5. Wenn primär ein „fetal distress" vorliegt, bestehen sehr hohe Laktatkonzentrationen beim Kinde, so daß hier auch nach langer Austreibungsperiode auf Grund des Konzentrationsgefälles eine „Infusionsazidose" nicht zustande kommen kann;
6. Ein Anstieg des mütterlichen arteriellen Laktatspiegels um ca 1.2 mmol/l begleitet die Einleitung jeder Vollnarkose. Seine Ursache wird diskutiert. Dieser Anstieg kann eine „Infusionsazidose" bei primären Sectiones hervorrufen, jedoch nicht bei Sectiones wegen „fetal distress".

Zwei praktische Schlußfolgerungen können aus unseren Beobachtungen gezogen werden:
– auch bei unauffälligem CTG-Verlauf sollte jede Geburt nach höchstens 8 Preßwehen operativ beendet werden, einerseits und
– eine möglichst kurze Hyperventilation sollte der Intubationsnarkose zur Sectio cesarea vorausgehen um eine metabolische „Infusionsazidose" zu vermeiden, andererseits.

Schlüsselwörter: „Fetal distress", Laktat, metabolische Azidose, materno-fetale Infusionsazidose, späte Austreibungsphase.

Résumé

Importance théorique et pratique de la détermination parallèle du lactate, du pH et des gaz du sang chez la mère et chez l'enfant au moment de l'accouchement.
Après des investigations préliminaires destinées à la définition exacte de la composante sanguine faisant l'objet de nos mesures, nous avons entrepris la détermination des concentrations de lactate dans le sang artériel maternel (L_M) ainsi que dans les vaisseaux du cordon ombilical (L_a, dans l'artère ombilicale et L_v, dans la veine ombilicale) à l'aide du Lactatanalysator 640 de la Maison Hoffmann La Roche au cours de 126 accouchements par voie basse et de 32 césariennes. Les résultats ont été rapportés d'une part à l'état du foetus à la naissance (données cardiotocographiques, scores d'APGAR), d'autre part à la durée de la phase d'expulsion et aux valeurs des gaz du sang relevées systématiquement.
Les conclusions suivantes en ont pu être tirées:
1. Il existe une relation étroite entre les niveaux maternel et foetal de lactate, en l'absence de souffrance foetale;
2. Le niveau maternal de lactate se trouve être en relation étroite avec la durée de l'expulsion;
3. Si l'expulsion est courte la différence de concentration de lactate reste dirigée des valeurs foetales vers les valeurs maternelles, ou bien l'on n'atteint que de faibles «infusions de lactate». Ainsi l'état foetal à la naissance ne peut en être compromis;
4. Si l'expulsion est longue le niveau de lactate maternel prédomine et il peut en résulter une acidose infusionnelle materno-foetale. Elle apparît déjà après 6 à 8 contractions expulsives et conduit, après environ 10 telles, à une souffrance foetale;
5. Lorsqu'une souffrance foetale préexiste, le niveau lactique de l'enfant est très élevé, de sorte qu'ici une «acidose infusionelle» ne peut apparaître, du fait des différences de concentration, même après une expulsion longue;
6. Une élévation du niveau maternel artériel de lactate d'environ 1.2 mmol/l accompagne l'induction de

chaque anésthésie générale. Sa cause fait l'object d'une discussion. De cette élévation peut résulter une «acidose infusionnelle» lors de césariennes réglées, mais pas au cours de césariennes pour souffrance foetale.

Deux conclusions pratique peuvent être déduites de nos observations:

- même lorsque le tracé cardiotocographique demeure normal, il convient de mettre terme à l'accouchement au moyen d'une extraction instrumentale, au bout de 8 contractions expulsives au plus, d'une part et
- l'intubation pour une césarienne doit être précédée d'une hyperventilation aussi brève que possible afin d'éviter une «acidose infusionnelle» métabolique, d'autre part.

Mots-clés: Acidose materno-foetale transmise, acidose métabolique, Lactate, période d'éxpulsion, souffrance foetale.

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Dr. med. D. Kalinkov
Universitäts-Frauenklinik
Pilgrimstein 3
D-3550 Marburg